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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Epileptic Patients Treated With Aureomycin

Clinical and Electroencephalographic Changes

F. W. STAMPS, M.D., E. L. GIBBS, and ERNST HAASE, M.D.
Chicago, Illinois

In any large group of patients with epilepsy approximately ten per cent will be found to have one or more of the following: a) a history of encephalitis antedating the seizures; b) a gradually progressive increase in clinical symptomatology and electroencephalographic abnormality; c) multiple focal electroencephalographic abnormalities, particularly those which over an extended period of time shift from one lobe to another. It was believed that a group selected according to these criteria might be expected to contain a high titer of cases in which the epilepsy was on an encephalitic basis.

Twenty-five cases were selected according to these criteria. They were cases with severe forms of clinical epilepsy, having daily or weekly seizures which could not be controlled either with maximal doses of commercially available anti-epileptic substances or a high dosage of experimental drugs. In six cases the seizures developed during or immediately after an encephalitis; in eleven they were presumably related to an acute febrile illness (two otitis media, two atypical pneumonia); in three they occurred after severe head trauma; in three they were associated with cerebral palsy which was present from birth, and in two cases there was no significant antecedent history. Examination of the spinal fluid in five patients including cultures, routine examination, animal innoculations, and serologic tests have all been negative.*

Only two cases had generalized convulsions alone; nine had convulsions and also short tonic atypical seizures associated with petit mal variant activity in the electroencephalogram; four had generalized convulsions and psychomotor seizures; seven had generalized convulsions and petit mal seizures. Two had only petit mal seizures, and one had only Jacksonian seizures. All had been having seizures for at least one year and one patient had them for eleven years. Twelve patients were under the age of ten; five were under twenty; eight were twenty years or older. Fifteen of the patients seemed to be gradually deteriorating, as evidenced by increasing retardation of speech and sluggish mentation.

Treatment consisted of adding Aureomycin 250 mgms every six hours to current anticonvulsant medication for children under fourteen; older children and adults were treated with 500 mgms every six hours. Treatment was usually carried out for five to six weeks. If no clinical or electroencephalographic response was noticed during the first week, the dosage was increased to 500 mgms every six hours for children and to 750 mgms every six hours for adults. In small children the first clinical signs of an Aureomycin effect were drowsiness and

From University of Illinois, College of Medicine, Illinois Neuropsychiatric Institute, Department of Psychiatry, Chicago, Illinois.

*These tests were made through the courtesy of Dr. Herman C. Mason of the Illinois Department of Public Welfare.

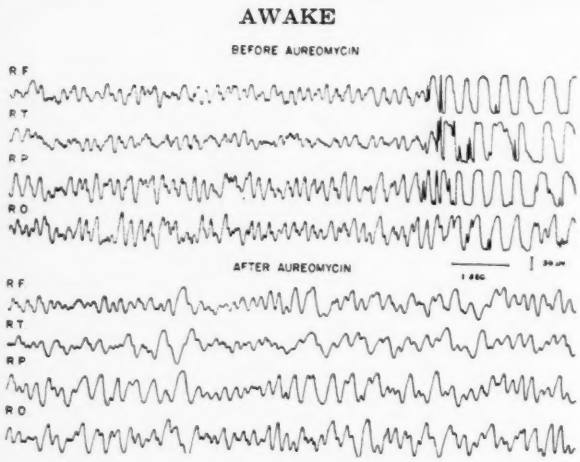

Fig. 1. Slowing of brain waves and disappearance of seizure activity.

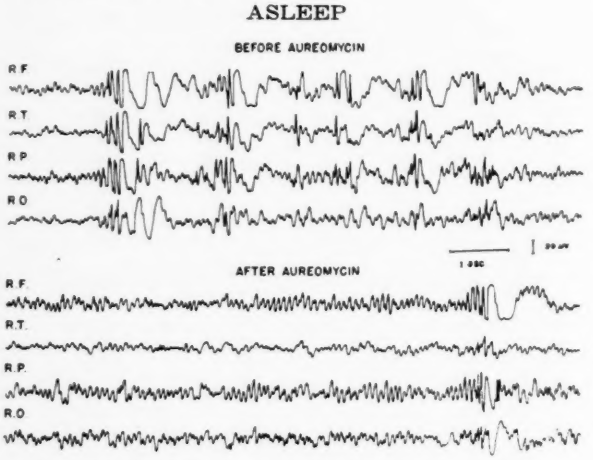

Fig. 2. Marked reduction in seizure activity.

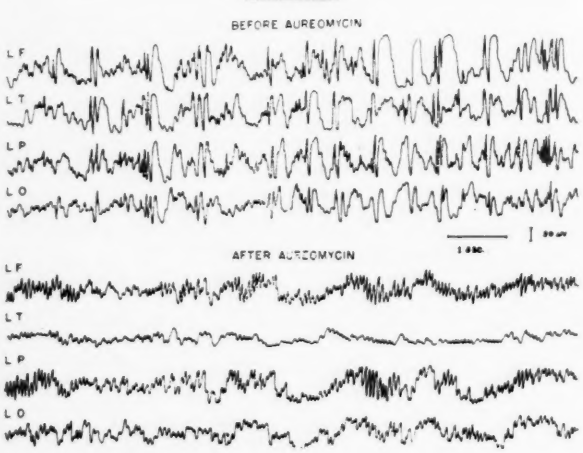

Fig. 3. Disappearance of seizure activity and normal sleep spindles evident.

ataxia. Evidently, these children could not well tolerate Aureomycin in addition to standard doses of anticonvulsant substances such as Phenobarbital, Dilantin, Mesantoin, and Paradione. When the *anticonvulsant*

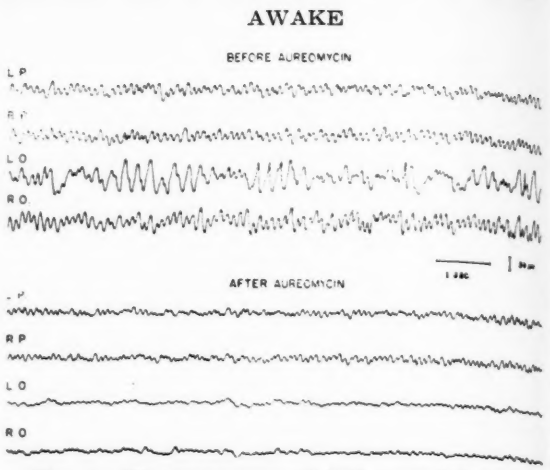

Fig. 4. Disappearance of left occipital focus of 4-6 per second slow activity and isolated spikes. Amplitude markedly reduced.

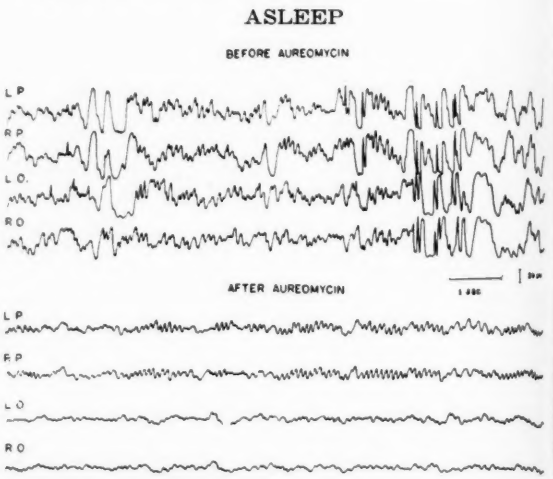

Fig. 5. Amplitude decreased and seizure discharges eliminated.

medication was reduced, the drowsiness and ataxia subsided and the Aureomycin was well tolerated. Children not receiving anticonvulsants did not develop drowsiness or ataxia. Slowing of the brain waves occurred concomitantly with the appearance of these symptoms and disappeared when they subsided. Figure 1 shows a slowing of from five to seven per second to three to seven per second in the awake record of a typical case. The slowing of the electroencephalogram was most noticeable in young children and not in adults. It usually appeared during the first week of treatment.

Ten of the twenty-five cases showed a lessening of seizure activity in the electroencephalogram. In seven cases with constant seizure activity in the electroencephalogram prior to the administration of Aureomycin, the seizure discharges disappeared

almost completely following the use of this drug. Figure 2 is a typical example of the lessening of seizure activity in one of these cases. Repeated records prior to treatment showed almost constant petit mal seizure activity and independent spiking; after treatment normal sleep frequencies appeared and during a forty-five minute recording only two very short bursts of seizure activity were obtained. Figure 3 is another striking example of how constant seizure activity disappeared following Aureomycin therapy. Many previous records had shown constant seizure activity and absence of sleep spindles. Normal spindles appeared after treatment. Figure 4 illustrates the improvement in the electroencephalogram of a patient with petit mal after two weeks' treatment with Aureomycin. In addition to much petit mal activity the awake electroencephalogram showed a focus of four to six per second slow activity and isolated spikes in the left occipital area. The seizure activity and focus disappeared completely and there was a pronounced reduction of amplitude especially in the occipital areas. Figure 5 shows a typical section of the sleep record of the same patient. Prior to treatment there were frequent short bursts of petit mal activity. After treatment no seizure activity was evident during one hour's recording. Sleep spindles were normal. The reduced amplitude seen in the waking record was still apparent. When the anticonvulsants were discontinued, the amplitude returned to normal.

Clinical improvement occurred in parallel to the disappearance of seizure activity from the electroencephalograms. Four patients have had no seizures for at least six weeks, despite gradual reduction in the dosage of anti-epileptic substances which they were taking prior to the use of Aureomycin. One of these four cases was a child who was previously having many petit mal seizures daily. She has not had a single attack since the second day she was placed on Aureomycin, and her electroencephalograms no longer show petit mal activity. Another child, age eight, who had been having many petit mal seizures daily for a year following an attack of "atypical pneumonia" showed a similar response. She had been unsuccessfully treated with Phenobarbital, Tridione, Paradione, and a number of anticonvulsants which are not yet commercially available. However, when after four weeks the Aureomycin was reduced to 500 mgms daily and the anticonvulsants were discontinued, a few fleeting petit mal attacks were observed in the course of a week. The previously normalized electroencephalogram showed one short burst of petit mal activity in an hour's recording. The Aureomycin dosage has been doubled and no seizures have been evident for the past week. Two cases have had no Aureomycin for two months and neither clinical nor electroencephalographic seizures have recurred. Another five cases have been greatly improved (clinical seizures reduced by 50 per cent) for at least one month.

The only drugs previously known to have a definitely normalizing effect on the electroencephalograms of patients with epilepsy were Tridione and Paradione, and this occurred only in patients with petit mal. Fortunately, Aureomycin seems to benefit a group of patients with a wide variety of electroencephalographic disorders whose seizures have been impossible to control previously. Nine cases which showed improvement electroencephalographically and clinically gave histories of an encephalitis or an acute febrile illness shortly antedating the onset of seizures. One cerebral palsy case with seizures from birth also improved. However, there were eight cases whose histories strongly suggested an encephalitic etiology who were not benefited. It is significant that none were helped whose seizures seemed due to head trauma or who gave no history of an associated febrile illness.

Most of the children in this series had behavior problems in addition to their seizures. Parents frequently reported an improvement in behavior shortly after the Aureomycin was started. In no case was it necessary to abandon treatment because of gastric disturbances. Several patients were temporarily relieved of chronic constipation and their appetites improved.

Although the evidence here suggests a chronic encephalitic process causing seizures, this study cannot rule out a metabolic effect of Aureomycin on the brain cells.

A much larger series of cases will be necessary in order to arrive at definite conclusions. Perhaps larger doses of the drug may be required in the older and more resistant cases. Other antibiotics should be tested for their effect on epileptic patients and on the electroencephalogram.

Summary and Conclusion: A preliminary report is given of the results obtained with Aureomycin in the treatment of twenty-five severe cases of epilepsy which had resisted all other types of medication. The longest period of followup is six months. However, in seven cases the improvement in the electroencephalograms and in the clinical condition of the patients were so dramatic that it seems likely this substance will be useful for the treatment of some types of epilepsy.

Electroconvulsive Therapy

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The search for methods of somatic treatment in psychiatry is by no means new. Aesculapius himself, is said to have prescribed alcohol for treatment of mental patients and Celsus in 14 A.D. made use of torture to restore his patients to sanity. A favorite treatment of that time was suddenly plunging the unsuspecting patient into cold water.

The electrical condenser was invented in 1746 and ten years later Richard Lovett reported success in the treatment of mental patients with electricity. Through the years, many and varied forms of somatic therapy found their way into psychiatric treatment. Emetics, purgatives, and castration were tried in turn and in our time Cotton advocated wholesale removal of teeth, tonsillectomy, and excision of a large part of the colon to remove foci of infection. When Wagner-Jauregg introduced malaria treatment for general paresis in 1917, it was to be expected that this procedure be tried on schizophrenia and other psychoses, as the mode of action of the fever was not understood at that time. Malaria fever, as well as typhoid and other heat producing agents, met with failure in these psychoses, and the search continued.

Many sedatives were used in the so-called sleep treatments. Opium had for many years been given for depression and in 1922, Klaesi introduced the use of barbiturates to produce prolonged narcosis, the immediate predecessor of shock treatment. Nitrogen, carbon dioxide, and continuous oxygen have all been administered to cause narcosis, with the hope that they might bring about improvement in mental patients. All of these brought more or less disappointing results, although both nitrous oxide and carbon dioxide are now being investigated, and more favorable results have been reported.

The present era of the so-called shock treatments began in 1933, when Sakel of Vienna reported on the treatment of schizophrenia by insulin-induced hypoglycemic coma. A year later in Budapest, Meduna caused convulsions in schizophrenics by intramuscular injections of camphor. Because the convulsive dose of camphor so nearly approached the lethal dose, he experimented with other convulsive drugs, and after discarding fifteen different preparations, he replaced the use of camphor with a synthetic, soluble preparation, which we now know as metrazol and known in England as cardiozol. He based his theory upon observation that schizophrenia is rarely seen in epilepsy and that the symptoms of schizophrenia may disappear after spontaneous convulsions, an assumption now considered by many to be not altogether correct.

The first suggestion that electrically induced convulsions be used in the treatment of mental disorders was made by Cerletti and Bini before the First International Meeting on Modern Treatment of Schizophrenia, held in Muensingen, Switzerland.

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in 1937. In 1938 they gave the first demonstration of this new therapeutic procedure in Rome, Italy.

Because of the extremely disagreeable sensations experienced by the patient, together with the large number of physical complications, the use of metrazol in convulsive therapy has become almost completely discarded. However, insulin and electrically induced convulsive treatment have continued to gain favor among psychiatrists and are known to be indispensable therapeutic measures in modern psychiatry.

With this brief historical background, we turn to consideration of electric convulsive therapy and its place in modern medicine.

The apparatus generally used to induce convulsions operates on 110 volts, 60 cycles, alternating current and consists basically of the variable transformer, Ohm volts and ampere meters, and an automatic timer. The electrodes are placed one on each of the patient's temples, which have first been smeared with a saline substance usually consisting of sodium chloride in a water soluble jelly. The initial application is usually of from 70 to 130 volts continuing for a period of from three-tenths to seven-tenths second and the combined voltage and time are known as the "dose." The usual milliampere range is between 200 and 1500.

A thorough physical examination of the patient prior to treatment is essential, not only to search for physical contraindications to the treatments, but, also, to have standards by which to compare physical findings following treatment. Authors differ widely in their precautions. Routine x-ray of the spine has been recommended to exclude patients with arthritis and to have a control when searching for compression fractures later. Such a film would also serve to reveal non-symptomatic vertebral compression received during previous treatments. It has also been suggested that chest x-rays be taken routinely to rule out heart disease and tuberculosis. An electrocardiogram is desirable but considered by many not indispensable. At the Medical College of Virginia, an E.K.G. is made on each patient about to receive electroconvulsive therapy. This examination is not so likely to reveal absolute contraindications as it is to show the physician cardiac defects requiring an extreme degree of precaution during the treatment. In such instances, a cardiologist is consulted before treatment is carried out.

The contraindications for convulsive therapy are not so numerous as formerly supposed and in each individual case the possible dangers must be carefully weighed against the probable benefits to the patient. It is kept in mind that frequently we are treating a patient whose behavior is so violent and disturbed that death is likely to result from sheer exhaustion if treatment is not administered as an emergency measure. Another may be so deeply depressed as to be constantly in danger of death by his own hands. In such cases we do not hesitate to begin treatment as quickly as possible, realizing well that some risk is involved when the patient has a known physical disease. Hypertension and myocardial disease are frequently considered contraindications. However, many disturbed patients with these conditions have been known to show rapid improvement after relief of anxiety, insomnia and generalized agitation. Most authors consider tuberculosis a contraindication; however, Hoch and Kalinowsky do not believe convulsive therapy harmful in that disease and think that it may help the psychotic tuberculosis patient by improving the mental condition. At the Medical College of Virginia, we do not delay giving electric convulsive treatment to acutely psychotic patients with pulmonary tuberculosis when the disturbed state of the patient is such as to prevent the proper rest and nutritional intake. As in the case of patients with cardiac complications, the shock treatment is not given until after the chest specialist has been consulted.

Apparently age is not a contraindication. Hemphill and Walter reported that they treated a child of three, and many patients in the eighth decade of life have been successfully treated, with benefit, for the affective disturbance accompanying senile mental states.

It is probable that complications do not arise from osteo-arthritis of the spine; however, decalcification, previous fractures and amputation stumps often present a serious

problem. The use of curare is strongly advised in these cases.

Brain tumor and increased intra-cranial pressure offer definite contraindications. A Parkinson's disease with depression may be treated without harm and convulsive therapy has become almost indispensable in controlling extreme overactivity and violence in the patient with general paresis. Hoch and Kalinowsky emphasize that cerebral hemorrhage following an epileptic attack is practically unknown, and that, therefore, the potential danger of cerebral hemorrhage should not be taken as a definite contraindication.

Other diseases contra-indicating electric convulsive therapy are exophthalmic goiter and acute infections of any kind, especially those involving the lungs or kidneys.

Kleinschmidt and his associates treated 825 patients with four fatalities, two of medical causes that could in no way be directly attributed to electroconvulsive therapy and one case was a suicide while under treatment. In our own series of more than 1,500 patients treated with shock therapy, we have had only one death. The patient did not die for several hours after the shock therapy but we attributed the death to the procedure and the patient showed considerable fatty degeneration of the heart on autopsy.

Traumatic complications have been greatly reduced since the electrically induced convulsion has replaced metrazol as a method of choice. However, complications are still frequent, especially compression fractures of vertebrae and joint dislocation. Preventive measures include hyper-extension of the spine, proper placement of the mouth gag and holding of the mandible, and proper immobilization of the large joints by manual pressure and fixation. A more recent innovation has been the use of curare to decrease the violence of muscle contraction during the convulsion. In many hospitals this drug is used routinely on all patients receiving electric shock treatment, while in others it is used only in the presence of known skeletal and muscular defects, backache, hernia, etc. On our service at the Medical College of Virginia Hospital, curare was used routinely for a time but it was discarded because it caused some respiratory embarrassment and we found that without its use there was a relatively low instance of traumatic complication. We felt we had worked out a satisfactory technique of prevention without adding the danger likely to be met with in the use of this drug. However, Bennett contends that the drug is perfectly safe in the hands of those who know how to use it and he uses it regularly.

Less serious complications are confusion, memory defect, amnesia of recall, and muscle soreness. These sometimes cause concern to the patient, who is usually relieved by an explanation that they are temporary and will disappear when treatment has been discontinued.

Before leaving the subject of contraindications and complications, it is well to consider shock treatment during pregnancy. Successful administration of electroconvulsive therapy during all months of pregnancy has been reported. Kent states "pregnancy should not necessarily be considered an indication to deprive a psychotic woman of needed shock therapy." Hoch and Kalinowsky are also in agreement with this opinion. Our experience is that its use in pregnancy is safe.

The procedure for administering the treatment is as follows: The patient's breakfast is withheld, as we prefer to give treatment at 8:00 A.M. and nausea is sometimes experienced if the procedure is carried out on a full stomach. Most usually the patient has been prepared psychologically for the treatment by having its need and probable benefits explained previously by the physician. This, of course, is sometimes impractical in severely psychotic patients. The patient remains dressed in whatever night clothes he has worn the night before, and in the case of out-patients, the outer garments are removed, and the treatment taken in the underclothing, unless a gown or pajamas have been provided. This is advisable because of the frequency of involuntary urination during the convulsion. The treatment is given upon a specially constructed, hard-top table, the surface of which is curved in order to produce hyperextension of the spine as the patient lies supine upon the table. This position has been found favorable as a

precaution against compression fractures of the vertebrae. In other hospitals where the treatment is given on a flat table or in bed, a pillow or a rolled blanket is placed under the back at the junction of the thoracic and lumbar segments of the spine. The saline substance is applied to the temples and the mouth gag, consisting of gauze rolled about a tongue depressor, is placed between the teeth in order to prevent injury to the tongue during the convulsion. Artificial dentures are removed beforehand.

It is essential that an adequate "team" be present. This consists of the physician who operates the treatment machine, the nurse specially trained to apply the electrodes and about four other persons to immobilize the patient by applying firm pressure at the shoulders, hips, and knees. This is a necessary precaution against bone dislocation and possible fracture.

Usually, the initial dose penetrates the convulsive threshold of the patient and there is a sudden tonus, followed by a longer period of clonus. Special care must be exercised to hold the mandible in place, as frequently the mouth opens with such violence as to cause dislocation of the mandibular temporal joint. After the clonic phase has ceased and respiration has returned to normal, the patient is placed on a bed and returned to the ward, where after a period of thirty minutes to one hour, he is usually able to get up, bathe, dress, and eat breakfast. There is almost always some degree of post-shock confusion and the patient should be observed by the nurse to prevent his getting lost on the ward or injuring himself. By noon the immediate confusion usually has disappeared.

Electroconvulsive treatment was first recommended for schizophrenia, and there were many reports of high rates of remission. However, in recent years, with more opportunity to observe schizophrenic patients over a prolonged period, there is an increasing number of reports of relapses. The group of schizophrenics responding best are those with a predominance of affective disorders —depression, catatonic, or manic-like excitement states. The best results have been observed in those cases with histories of acute onset and presenting strong affective reactions and in individuals with good previous social adjustment. The paranoid types are known to be more resistant to treatment, and a large number of convulsions are usually indicated, and, even then, the results are frequently unsatisfactory.

Electric shock treatment is almost specific for the affective psychoses. Bennett reports uniform results of 80 to 90 percent full or social recoveries from all types of depressive reactions, with best results seen in involutional or pre-senile depressions. These were formerly considered the most resistant to therapy and no previous method was known to terminate the condition except patient supportive therapy while awaiting spontaneous recovery.

Patients with manic excitement states respond almost as well as the depressions but usually require a greater number of treatments and are more likely to undergo relapses. The depressions frequently show marked improvement after five or six treatments, whereas the manic is likely to require twelve to fifteen.

Convulsive therapy is not generally indicated in the psychoneuroses and should never be used as a substitute for sound psychotherapy. However, many psychoneurotics are encountered in whom the predominating symptom complex is centered about the presence of a severe anxiety state. These patients frequently respond to a small number of convulsions, with noticeable relief of symptoms, becoming, therefore, much more receptive to a series of psychotherapeutic interviews. Reactive depression, which is considered a neurotic depression, responds readily to shock therapy. Although Bennett believes that obsessive compulsive neuroses do not respond well unless secondary to depression, it has been our experience that these cases often receive some relief by confusing doses of electroconvulsive therapy. This is done by giving the patient one treatment daily, until a marked organic confusion state is brought out.

Early in our use of electroconvulsive therapy we were greatly disturbed when a patient developed mental confusion and amnesia of recall. We soon learned, however, that these two conditions, either singularly or together, would rapidly clear up when the

treatment was discontinued. In fact, it is the belief of some that the creation of a confusional state by shock therapy is desirable. As a matter of fact, we not infrequently deliberately create this condition in certain apparently intractable cases of depression, and also in those who show early response to electric shock with short periods of remission between attacks.

The conventional frequency of treatment is every other day, or more specifically, three times weekly, but it is occasionally necessary to increase the frequency, sometimes to daily doses for several days and often two, and sometimes three, separate individual shocks in a day. This latter procedure is necessary in the very acutely disturbed patients and it is remarkable how quickly these combative, assaultive, maniacal individuals can be quieted and be tractable in a few days and be able to be moved to an open ward. The disturbed section of the Psychiatric Division of our hospital is now, due to this procedure, only partly full. Continuous tubs and similar hydrotherapeutic procedures have been almost entirely discarded and for this reason it is seldom necessary to use drug therapy.

The mode of action of electroconvulsive therapy is not completely understood. Gordon collected 50 theories which he divided into somatogenic and psychogenic effects. Under the somatogenic theories, he mentions (1) a weakening of associated traits; (2) alteration of brain metabolism; (3) effect on nerve cell membranes; (4) production of capillary spasms with reduction of cerebral activity. As psychogenic effects, he points out (1) the mobilization of drives of self-preservation; (2) unconscious feelings of dying and resurrection; (3) release of inhibitions; (4) atonement of guilt.

It has been found that the electroencephalographic changes are in direct proportion to the number and frequency of treatments and usually the electroencephalogram shows a return to normal in about two weeks after the last treatment is given.

Bennett believes that it is the organic confusional state which causes the improvement in the affective disorders. The patient forgets his problems and the self-consciousness and obsessive thinking are broken up. On the other hand, we have observed marked improvement in patients with affective disorders after only two or three treatments and showing almost no confusion. Actually, whether or not the confusion from the treatment is necessary for improvement awaits further study.

Electric convulsive treatment is frequently given in combination with subshock insulin. The electric shock treatment is administered while the patient is still in a state of semi-stupor, and therefore, free of apprehension and anxiety in anticipation of the treatment. Intravenous amytal in doses from 3¾ to 7½ grains given before the procedure reduces the blood pressure and apprehension and lessens the fear. This drug has proven exceedingly useful in giving us a method by which to treat a patient without his becoming aware of ever having received the treatment. This method is often considered advisable in treating delicate, neurotic, or depressed patients, showing extreme fear of the therapy. After deep sedation an increased "dosage" is required to break the patient's convulsive threshold and additional precautions must be taken to watch for and combat prolonged respiratory arrest.

Because of severe post-convulsive confusion and combativeness in certain patients, it is sometimes advisable to give 3¾ to 7½ grains intravenous sodium amytal following the treatment. This is given after the state of clonus has ceased and respiration resumed, but before the patient has time to begin thrashing about or crying out. Giving the drug at this time to prevent post-convulsive agitation rather than before the treatment, has the advantage of not raising the convulsive threshold and not causing respiratory embarrassment.

Convulsive therapy may be given on an out-patient basis. Such a plan is especially useful in treating certain selected patients who have undergone a period of intensive therapy in the hospital and whom we feel may be maintained in a satisfactory state of social remission on one treatment per week. If the patient continues to get along well on one weekly treatment, the frequency may be extended to once every ten days or two weeks. Out-patient treatment also may be

administered to certain depressives and neurotics.

We seldom give out-patient treatment to anyone who has not first been hospitalized. In this way the patient is assured of a more thorough physical examination and personality study than would be possible if he came directly to the treatment room from the office or his home. Furthermore, we have observed that patients with illnesses for which shock treatment is indicated do much better and show more rapid improvement if separated from family and friends for at least the first part of the therapeutic course.

In the final analysis, the true value of any form of therapy is its effect upon the prognosis of the illnesses in which it is applied. Unmistakable evidence of social recovery and, in many cases, complete recovery, has been observed in all varieties of affective disorders treated with electroconvulsive therapy. In the schizophrenias almost invariably the acute episodes are greatly reduced and usually a social recovery is attained if the patient is in the early stages of the disease process. However, we know now from our experience, that this illness frequently progresses through a course of many exacerbations and remissions to a stage which finally is affected little or not at all by electric shock. In spite of some failures it is the general opinion of psychiatrists that the shock therapies point to an improved over-all prognosis and that prognosis is improved by shock treatment in those cases in which it is already favorable. It cannot be denied that, although electroconvulsive therapy is not a cure for schizophrenia, it does greatly facilitate the care of the patient while in the hospital, shortening his stay and increasing the number of social remissions.

Electric shock is by no means a panacea for all nervous and mental illnesses. It is certainly not indicated in all neuropsychiatric disorders but we do know that it is most useful in the affective mental states and is almost a specific in the involutional depressions. Whether electric shock ever restored anyone to a normal mental state who might not have obtained this result from other procedures is not known, but in our thirty years' experience more people with mental illnesses are getting well and in a much shorter time since the advent of the modern physical therapies.

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Review of Certain Lobotomy Processes

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The following report was prepared in an effort to provide an orientation to the essential psychological processes consequent upon lobotomy operations. There has been no attempt to secure an exhaustive survey of psychosurgery problems. Only some of the more important experimental findings are discussed, together with the theoretical formulations involved.

Conflicting test results occur in lobotomy literature, depending usually on the type of test used and the abilities measured. Within the purview of particular kinds of test criteria, however, there is a general agreement on the type of changes that occur in lobotomy operations. It is proposed by Babcock[3] that a single test can't be used to disclose significant results, but that a comprehensive battery is necessary to reveal specific strengths and weaknesses.

In most studies there is no reduction or increase in intelligence scores as a result of lobotomy,[6, 21, 30, 39] either immediately following surgery or later. According to Halstead,[20] even tests of "biological intelligence" show no consistent picture. Standard intelligence tests used in lobotomy experiments include the Wechsler-Bellevue, Stanford-Binet, Kuhlmann-Anderson, and the Army Alpha.

The results of memory and orientation tests are more variable. Acherly[1] found no evidence of recent or remote memory impairment but notes that there is a marked concentration on only one item at a time. This seems to be a characteristic post-lobotomy symptom. Crandell[6] reports no significant changes in learning, memory or abstraction abilities in his experiments. Babcock[3] found that the memory span was shorter following surgery.

Goldstein[17] names as the oustanding lobotomy change an impairment of abstract attitude and explains that patients' performance varies as the task is embedded in a concrete or abstract situation.[18] He argues that memory failures are not caused by impaired mental content but from a faulty attitude or approach (to the Goldstein-Scherrer test of abstraction). Lobotomy patients are satisfied with an inferior achievement and their basic change is not in any one field or performance but in their total behavior.

Friedlander,[16] while he undertook no pretesting, found significant mental impairment manifested in the test interrelationships. His more specialized tests include Kohs Blocks, the Porteus Maze, the Vigotsky Block Test, and the Babcock-Levy Test.

Various performance tests have been used to measure lobotomy changes. No appreciable changes in achievement were disclosed on the Grace Arthur Test on pre- and postoperative evaluations.[1] There was no hesitation in executing decisions once made, but there was a tendency to quantity production rather than quality production.

The Porteus Maze has frequently been reported in the lobotomy research literature.[38, 39, 40, 41] Surgery accounts for a drop of about one and one-half years in average test age, although much of this decline is recoverable with time. There is considerable impairment with regard to planning, foresight and executive ability. The loss in planning seems to be permanent. It is interesting to observe that test impairment is seen in improved as well as unimproved patients. As the time interval after surgery increases, there is a greater tendency for the patient to make stereotyped or repeated errors. In this test also, it is evident that the patients have difficulty in dividing their attention. Attention difficulty and poor planning is also verified in the results reported by Robinson,[44] using a large battery of tests including Deliberation Tests and the Will-Temperament Test; in these two tests the lobotomy patients did less well than a control group. He also used the Hunt-Minnesota Test of Organic Brain Damage and found that retroactive-inhibition was not increased.

Personality tests have been mainly limited to the Rorschach. According to Hutton,[27] lobotomized patients show a reduction in originality and creativity. Crandell[6] and Harrower-Erickson[21] report no significant personality changes on the Rorschach. Kisker[28] indicates that Rorschach improvement may or may not run parallel to clinical improvement. An investigation by Van Waters[47] reveals that the Rorschach records of schizophrenic lobotomy patients is consistent with a schizophrenic classification despite surgery, and that organic signs are secondary.

Numerous behavioral studies of lobotomy patients have been done. The results of most of them are in substantial agreement with those of Birch.[4] He lists four prominent effects of cutting the frontothalamic fibers: (1) Diminished self-consciousness, (2) Diminished affect, (3) Diminished worry over guilt, sin, etc., and (4) Reduction of hypochondriacal ideas. The decrease in self-consciousness eventuates in a lack of critical insight and a certainty of being right, according to Babcock.[3] Acherly[1] adds that there may be a tendency to purposeful over-activity, since there is more energy and less fatigue (probably also attributable to a diminution in psychological awareness). This may be punctuated with periods of unusually passive behavior. When emotional flare-ups occur, recovery is quick. He notes the presence of loquaciousness at times, although without any pressure of speech or flight of ideas. Mood reactions become stabilized, although more self-assertion is noticed—along with occasional vulgarity. As expressed by Frankl,[13] self-esteem is high. He concludes that definite symptoms are most apparent immediately following surgery, and gradually disappear. There may be a ravenous appetite, distortions of memory and retention, apathy and laziness, irritability and lack of emotional inhibition. Freeman[14] lists as possible unfavorable sequalae, persistent inertia, convulsive seizures, incontinence, and aggressive misbehavior.

Hutton[26] agrees that lobotomy patients have a shallow emotional life, although they may react normally to immediate situations. Liebert[29] emphasizes that some patients improve behaviorally and not ideationally. The occurrence of a mild hypersexuality is reported for some patients, both by McKensie[30] and Acherly[1]; this may be denied by patients themselves. Goldstein[19] believes that lobotomy patients lack initiative and are impaired in voluntary shifting and choice. Myerson[33] reports that old people with chronic depressions are not to be regarded as hopeless as far as lobotomy treatment is concerned. He studied four women in their declining years who were chronically ill and depressed. There was a radical improvement with surgery and alleviation of tension and distress.

Crandell[6] believes that the same basic personality traits persist despite lobotomy, although overt psychotic symptoms are usually removed. His impression is that as dissociation of affect from ideation increases, prognosis with operation becomes less favorable. This concurs with Frankl's opinion[13] that the hebephrenic syndrome is too similar to post-lobotomy symptoms for improvements to occur.

Draper[9] has outlined a psychic tension syndrome as the chief criterion for lobotomy. This is found in patients whose emotions are largely concerned with themselves. The most favorable results should occur in obsessive, hypochondriacal, intractable psychosomatic conditions and agitated depressions. The main improvement in these patients is that they become more manageable and make a better institutional adjustment. Dibdens[8] reports that obsessive patients experience a loss of their obsessions, and a reduction in tension and anxiety; they still remain punctual and meticulous, but are more happy and cheerful.

Freeman and Watts[15] expect a good prognosis in anxiety neurotics and neurotics with hysterical conversions. Schizophrenic patients are favorable lobotomy prospects if they are excited, resistive, assaultive and disturbed. Quiet, deteriorated patients usually remain unchanged. Alcoholic, psychopathic, criminal, and patients with organic brain disease seldom benefit from frontal surgery.

Frankl[13] conducted a study of 68 patients with a view toward evaluating their community adjustment. More than 50 per cent

recovered sufficiently to earn a living and more than 75 per cent became gainfully employed. Most of them took part in community life but made no new friends. They tended to be self-willed, domineering and lacking in the qualities of affection and consideration, although there was no anti-social behavior. As a whole they were restless, showed frequent interest and job changes and lacked discrimination in money matters. He observed a greater occupational deterioration in schizophrenics than among other patients.

Four favorable prognostic criteria for lobotomy candidates have been proposed by Hurt[25]: (1) Acute onset, (2) previous personality reasonably well-balanced and integrated, (3) under middle age, and (4) not chronic. Liebert[29] also feels that there is a better prognosis for those patients who have been ill for less than five years. Rietman[42] argues that only patients with well-preserved personalities should be operated. He adds that their likelihood of being discharged from the hospital is better before middle-age. He offers that prognosis is not dependent on chronicity if pre-operative personality is relatively well-preserved. In another study of 59 female schizophrenics, Rietman[43] found 58 per cent of them showed some improvement. He felt that one of the main changes was that they became more accessible to psychotherapy.

Parker,[34] in an investigation of 100 female lobotomy patients, doubts if lobotomy is more successful than other forms of therapy in paranoid schizophrenia. He suggests an early operation before other methods of treatment have been instituted in cases of patients with long-standing poor social adjustment.

Coheen[5] recommends a continued program of rehabilitation in a favorable and interested environment as a factor aiding in readjustment. The cases of successful adjustment in his study were often preceded by a long period of organic sluggishness and retardation.

Acherly[1] studied the behavior of a lobotomy patient over an extended period of time. Four months after her lobotomy she was euphoric, over-active, playful and childish. She tried to dictate to her husband and family and was extravagant in her handling of money. Most of these symptoms disappeared in six months but a mild feeling of well being and youthfulness persisted for a year. During the second year this youthfulness faded. She became relatively responsive but had little concern about things remote. There was an inability to think about more than one thing at a time.

Acherly[1] feels the strong concentration on only one thing at a time might be due to an increased power of concentration or to a lack of ability. In any event, it supports the idea that frontal lobes enable us to become aware of an increasing number of simultaneous impressions.

Edwards[10] concedes that we don't know what lobotomy does. It is conceivable it may produce its effect in a general way by virtue of being a violent and determined attack in a highly specialized region, as contrasted with electric shock therapy, for example.

Freeman and Watts[14] propose that the frontal lobes are concerned with a projection of the total individual into the future and implement a high order, intellectual function. They[15] advance the hypothesis that frontal lobe function is specific and that it is concerned with foresight and insight.

Goldstein[17] also emphasizes the molar approach, asserting that the presence or absence of emotional expression corresponds to the entire behavior in a given situation. Emotional behavior is inseparable from the rest of the person's behavior and the basic change is not a change in any one field of performance but a change in total behavior. The change that occurs seems to be a change in abstract attitude resulting in a decreased capacity for maintaining a deliberate direction of thought and movement.

Robinson[44] states that the frontal area severs the function of prolonged attention, thus explaining the reason why it relieves obsessive tension.

Friedlander[16] speculates that perhaps the functional psychosis before lobotomy is replaced by an organic psychosis after lobotomy. This treatment, while epigrammatic, doesn't coincide with the data accumulated by other investigators. In previously cited studies we have noted that basic personality

patterns remain essentially unchanged after lobotomies and that organic complications are secondary.

Summary

There is essential agreement upon the type of psychological changes that occur as a result of lobotomy operations. These changes have been measured by means of psychological tests and clinical studies. Conflicting experimental results depend usually on the type of criteria used and the differential abilities that are evaluated. A single test, e.g., can't be employed to disclose significant results, but a comprehensive battery is necessary to reveal specific strengths and weaknesses.

In most studies there is no reduction or increase in intelligence scores. Memory and orientation functions are more variable, but impairment, when noticed, seems to decrease with time. A partial explanation for this variability seems to reside in lobotomy patients' difficulty in dividing their attention. There is a marked concentration on one thing at a time, with a considerable impairment in planning, foresight and executive ability. The loss in planning seems to be permanent and occurs in improved as well as unimproved patients. This would seem to point to an organic impotence. It is suggested by at least one author that memory failures are not caused by impaired mental content, but from a faulty abstract attitude or approach; the basic change would then be reflected as a change in total behavior rather than a change in any one area or performance.

Personality changes would seem to be inconsiderable, and it is proposed that basic personality changes may or may not run parallel to clinical improvement. Some patients improve behaviorally and not ideationally. In the records of schizophrenics, e.g., their post-operative behavior is usually consistent with the schizophrenic classification despite surgery. In a study of 68 patients who were returned to normal community life, it was shown that they tended to be self-willed, domineering and lacking in the qualities of affection and consideration.

Two prominent features of lobotomy are a diminished self-consciousness and diminished affect. The decrease in self-consciousness eventuates in a lack of critical insight and a certainty of being right. Mood reactions tend to become stabilized, although more self-assertion may be noticed.

The chief criterion for lobotomy seems to be a psychic tension syndrome. As dissociation of affect from ideation increases, the prognosis with operation becomes less favorable. The most favorable results should occur in patients with obsessions, hypochondriacal complaints, intractable psychosomatic conditions and depressive states. A good prognosis may also be expected in anxiety neurotics and neurotics with hysterical conversions. Schizophrenic candidates are most favorable if they are excited, resistive, assaultive and disturbed.

The frontal lobes seem to be concerned with a projection of the total individual into the future. Experimental work appears to support the idea that the frontal lobes enable us to become aware of an increasing number of simultaneous impressions. After lobotomy the ability for sustained attention becomes impaired; this helps to explain why obsessive tension is relieved since the patients' anticipatory capacities are diminished.

In general, the main improvement that can be expected from lobotomy is that the patients become more manageable and make a better institutional adjustment. They also become more accessible to psychotherapy. A continued program of rehabilitation in a favorable and interested environment is suggested as an important factor aiding in readjustment.

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Hypnosis and Productive Orientation

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Erich Fromm commented in his book, *Man For Himself, An Inquiry Into the Psychology of Ethics,* "An example, though an extreme one, of non-productive activity is the activity of a person under hypnosis. The person in a deep hypnotic trance may have his eyes open, may walk, talk, and do things; 'he acts.' The general definition of activity would apply to him, since energy is spent and some change brought about. But if we consider the particular character and quality of this activity, we find that it is not really the hypnotized person who is the actor, but the hypnotist who, by means of his suggestions, acts through him. While the hypnotic trance is an artificial state, it is an extreme but characteristic example of a situation in which a person can be active and yet not be the true actor, his activity resulting from compelling forces over which he has no control."

The quoted paragraph is a mere fragment extracted from a volume replete with interest and merit. As part of the whole it is of relatively little significance. Intrinsically, however, it is deserving of attention and in its relation to psychotherapy in general it is important.

The observation is part of a section called "The Productive Orientation." As such, it possesses a fundamental relationship to the concept of productiveness in treatment, with specific reference to the use of hypnosis in treatment. The implications of the view expressed are obvious because of Fromm's participation in psychoanalytic work. In this discussion, however, the focus is not on the author himself. Rather, it is on the nature of the observation owing to its widespread acceptance without adequate scrutiny on the basis of actual experience.

The quoted comment is only partially correct. In addition, however, it is significantly incorrect. The extent to which it is correct is so minor, that the paragraph could have been omitted completely without loss. The degree to which it is incorrect may have justified alteration. In such altered form, especially if related specifically to therapy, it could have furthered the fruitful discussion of examples of productiveness in its most positive aspects.

It is true that under hypnosis a person may have his eyes open, may walk, talk, and do things. Since he also does this without hypnosis, the variable then is the existence of the hypnotic state. The walking, talking, and doing may be part of a relatively simple conversational situation in hypnosis. As such it has little bearing on productiveness. If what is said and done begins to transcend what is ordinarily experienced in the non-hypnotic state so that it becomes somewhat dramatic, then it is reasonably correct to say that the hypnotist by means of his suggestions is acting through the subject. Even then, the subject will incorporate into his behavior, his peculiar personality characteristics lending modification to the nature of the acts requested of him. If sado-masochistic components are involved in the hypnotic relationship, they simply demonstrate a more dramatic illustration of such as are also found in non-hypnotic situations. Thus far, the meaningfulness of these facts for a discussion of productiveness is minor.

When it is said that "An example, though an extreme one, of nonproductive activity is the activity of a person under hypnosis," it may also be said that an example of productive activity is that of a person under hypnosis. This is of relatively greater interest from a scientific point of view, and it is of unquestionable significance insofar as therapy is concerned. As a matter of fact, at times such an example may be equally "extreme" if not more so.

When a patient in hypnosis is asked to dream and he proceeds to do so, he is acting in accordance with a suggestion. If he supplies the dream and nothing more he is not productive. Likewise, a patient in analysis when asked to present his dreams may present them as a gift without using them constructively. He too is not productive. When

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in hypnoanalysis a patient is told that he will have a dream which he will be able to interpret, that this dream will assist in the clarification of a particular problem as a result of which he will find himself capable of making a significant decision, and the patient follows through effectively, that person is functioning productively. The events relate to a suggestion given him, but this stimulus has as its response something which is, in fact, productive. He is utilizing his own resourses and potentialities. If, despite the nature of the suggestion, a dream were supplied and nothing more, there would be no productiveness. This would be the equivalent of the first example given above. If a patient is told in hypnosis that he will have a series of dreams which will help him to understand the nature of his relationship with another individual, and if the patient responds effectively with emerging recognition of a dependency relationship, for example, he is being productive. Of course the additional need to work through the problem and alter the relationship is required and this also involves productiveness. The process, however, may well be effected in the hypnoanalytic as well as non-hypnotic situation. The comparative merits are of interest but not directly pertinent here.

The hypermnesia often encountered in hypnosis is well known. In certain instances the recall of buried memories is accompanied by effective spontaneous integration of the data elicited with various insights available to the patient at the time and with consequent beneficial therapeutic effect. Additional insights may be crystallized thereby. The important point is that the resources and potentialities of the patient find expression following the initial therapeutic stimulus and emotional growth ensues. It is true that a "suggestion" is frequently involved to begin with, but this may more propertly be thought of as a guiding note in the same way that it may be employed in non-hypnotic procedures. Just as a "basic rule" may be employed in analytic treatment to create a special type of therapeutic situation, so too there are hypnotic techniques of various kinds and they create special situations also.

An hypnotic regression to an earlier age level with the re-living of certain experiences need not be productive. Frequently, however, the outcome demonstrates productiveness as the result of the integration of data evoked with various aspects of personality functioning which furthers insight and progress. This applies also to a variety of techniques which may be incorporated into hypnosis such as free asscciation, varieties of hypnotic visual imagery, automatic writing, hallucinatory experiences and others.[2-7] These methods, in addition to their use for furthering productive activity as described, may be employed also in the analysis of the transference relationship. Hypnotic techniques may also be employed to assist in the analysis of the hypnotic transference specifically or for further clarification of the meaning of the hypnotic state for the person involved.[8-12]

In view of the fact that behavior in hypnosis, as a result of the use of special techniques, can signify a process of personality growth involving the potentialities of the patient, then hypnosis may be used as an example of productive activity. This would be accurate and important. Since there are so many opportunities for further investigation of hypnosis within the framework of therapy, this aspect of hypnotic behavior is deserving of greater stress.

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A Statistical Analysis of 400 Sodium Pentothal Interviews of 125 Individual Cases

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The point has been reached in this series of articles on sodium pentothal, having appeared periodically in this journal,[1-7] to be able to report with statistical significance upon 400 sodium pentothal interviews performed upon 125 individuals with favorable improvement.

The data presented represents a basic interpretation of the statistical analysis and also the statistics as related to psychiatric diagnosis without offering any clinical evaluation; the exact cases will be presented in two subsequent follow-up studies on anxiety states and heterogeneous conditions, offering their own details of the psychiatric phases of clinical significance.

In the years 1947 to 1949 inclusive, 125 individuals were subjected to 400 sodium pentothal interviews. Completely unrelated to the psychiatric diagnosis the statistical breakdown of the number of individuals subjected to the number of interviews is as follows: 14 individuals had 1 interview, totaling 14; 36 individuals had 2 interviews, totaling 72; 32 individuals had 3 interviews, totaling 96; 17 individuals had 4 interviews, totaling 68; 12 individuals had 5 interviews, totaling 60; 8 individuals had 6 interviews, totaling 48; and 6 individuals had 7 interviews, totaling 42.

To divide the above statistics according to psychiatric diagnosis, 85 of the cases were of the anxiety state and had 275 interviews with the following breakdown: 10 individuals had 1 interview, totaling 10; 23 individuals had 2 interviews, totaling 46; 22 individuals had 3 interviews, totaling 66; 13 individuals had 4 interviews, totaling 52; 6 individuals had 5 interviews, totaling 30; 6 individuals had 6 interviews, totaling 36; and 5 individuals had 7 interviews, totaling 35.

The remaining 40 individuals received 125 interviews listed under the classification of heterogeneous cases. In relation to this group, it is necessary to record the statistics from two points of view: namely, the actual numerical figure expressing the number of interviews per patient unrelated to subdivision of clinical diagnosis, as was able to be done for a homogeneous group as the anxiety states above; as well as the statistical breakdown according to each of the separate categories of the various conditions.

Thus, the statistical analysis of the heterogeneous cases without subdivision of clinical classification is as follows: 4 individuals had 1 interview, totaling 4; 13 individuals had 2 interviews, totaling 26; 10 individuals had 3 interviews, totaling 30; 4 individuals had 4 interviews, totaling 16; 6 individuals had 5 interviews, totaling 30; 2 individuals had 6 interviews, totaling 12; and 1 individual had 7 interviews, totaling 7.

In relation to the clinical subdivision of the heterogeneous cases, we have divided the psychotic states into two categories; namely, pre-hospital and office, and post-hospital.

PRE-HOSPITAL AND OFFICE		22
Schizophrenia		14
Hospital	11	
Ambulatory	3	
Depression		8
Hospital	5	
Ambulatory	3	
POST-HOSPITAL		5
Schizophrenia	2	
Depression	3	

The first category includes schizophrenia, both the ambulatory type and cases previous to ultimately obtained hospital care; as well as depressions previous to hospital care and those receiving no hospital care at all. This category totals 22 individuals with the following breakdown: there were 14 schizophrenics, of which 3 were the ambulatory type, 2 of whom received 2 interviews and 1 received 4 interviews, totaling 8; 11 were of the pre-hospital type, ultimately requiring a course of electric-insulin shock therapy, of which 2 individuals received 1 interview, 1 individual received 2 interviews, 5 individuals received 3 interviews, and 3 individuals received 5 interviews, totaling 34; there

were 3 ambulatory depressions receiving 2, 3 and 6 interviews, totaling 11; and, there were 5 depressions hospitalized, of which previous to hospitalization 1 individual received 1 interview, 1 individual received 2 interviews, 2 individuals received 3 interviews each, and 1 individual received 4 interviews, totaling 13.

Of the post-hospital cases, there were 3 depressions 2 of whom received 2 interviews each, and 1 individual received 5 interviews, totaling 9. There were 2 schizophrenics in this group that received 3 and 4 interviews, totaling 7.

Some definite improvement was noted in 7 cases of alcoholism, of which 3 individuals received 2 interviews, 1 individual received 4 interviews, 1 individual received 5 interviews, 1 individual received 6 interviews, and 1 individual received 7 interviews, totaling 28. Following their original report on the use of sodium pentothal in alcoholism, Lemere and O'Hollaren [8-9] report even more favorable results in their later reports.

In the field of child psychiatry, we subjected 1 child to 1 pentothal interview with sensational results. The clinical details of this case will be presented in the follow-up study on heterogeneous cases. Cornil and Ollivier[10-11] have published several reports offering favorable data in the use of the sodium pentothal interview in child psychiatry.

The final group of cases in this series, the clinical details of which will appear in a subsequent paper, represents isolated cases in which the use of sodium pentothal interviews aided materially in conjunction with associated therapies. This category includes a case of stuttering subjected to 2 interviews, a case of neuro-dermatitis subjected to 3 interviews, a case of drug addiction with anxiety subjected to 2 interviews, a psychopathic personality, and a case of compulsive-obsessive neurosis subjected to 5 interviews, totaling 14.

Summary and Conclusions

1. We have presented the statistical analysis of 400 sodium pentothal interviews performed on 125 individuals.

2. The sample is now sufficiently large to attach statistical significance to the value of the results of the technique.

3. The psychiatric details of the cases shall be described in subsequent papers of this series.

4. Above all, the favorable responses recorded above over a period of 36 months warrants further investigation, both in respect to increasing the size of the sample, as well as extending the period of time involved.

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Diabetic Dorsal Sclerosis of the Spinal Cord

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Neuropathy in diabetic patients is conceded to be more common than available reports would indicate. Rundles[1] studied 3000 diabetic patients in the medical wards of the University Hospital at Ann Arbor, Michigan, in the decade from 1936 to 1946 and a diagnosis of diabetic neuropathy was made in 125, an incidence of 4.17%, or almost one out of every twenty patients. This neuropathy is frequently diffuse, disseminated, and often presents a bizarre picture, with predominant symptoms either of cerebral origin or related to the spinal cord, or the result of changes in the peripheral nerves.[2] This frequently observed fact means that practically any neurologic syndrome that has been described may occur during the diabetic lifetime. It has been my personal observation that most physicians think primarily and only of peripheral neuritis, the commonest manifestation of neuropathy in diabetes. Perhaps this is because this latter is the only picture stressed in medical school.

Little is known concerning the exact pathology of nervous tissue degeneration in diabetes and far less of its pathologic physiology and etiology.[3] In early stages, whatever the pathology at the time, the changes seem to be reversible, and control of the mismanaged or uncontrolled diabetic, in which most of these complications occur, seems to offer the best chance of recovery.[1]

Diabetic neuropathy has been considered by some to be the result of degenerative changes resulting from the disease of the arterial supply,[4] by others as nutritional deficiencies. A study of 100 cases of diabetic neuropathy by Rudy and Epstein in Boston[5] led these authors to the conclusion that the wide-spread nervous system damage observed was due to profound biochemical disturbance in diabetes which favored vitamin deficiency, not due to poor dietary intake. Notorious failure to reverse these changes with huge doses of vitamins in many patients seems to invalidate this concept. These authors' success with vitamin therapy has not been duplicated by others. However, in those patients who do recover rapidly, as Gregory and Lindley[6] point out, it is difficult to believe that the specific pathologic lesion is a degenerative one. These authors also emphasize the varied and widespread portions of the nervous system involved, due not to high blood sugar itself but associated somehow with the uncontrolled diabetic process. They offer an attractive theory that with carbohydrate deprivation perhaps excessive oxidation of fat leads to demyelinization. There is little valid evidence to support the belief that any member of the B fraction, or insulin, is a responsible etiologic factor.

Regardless of our lack of knowledge concerning precise pathologic and etiologic factors in diabetic nervous system disease, there seems to be general agreement that to prevent these changes adequate control of the diabetic is desirable. Here is where prevention is better than cure. And it seems that control to the point of merely avoiding acidosis is not sufficient. As far as treatment is concerned, most agree with Marble at the Baker Clinic in Boston,[7] that unless the diabetes is brought and maintained under meticulous control for a prolonged period, all other attempts at treatment will fail. In the past few years, the tocopherols or vitamin E have been used with increasing frequency,[8] but fuller evaluation of this therapeutic agent in neuromuscular diseases is needed. Along with adequate control of the diabetes, most therapeutic aims today include ample vitamin intake.[9]

With this brief introduction of the subject of nervous tissue change in diabetes, this writer reports two cases of so-called diabetic dorsal sclerosis of the spinal cord, often referred to as pseudo-tabes. These cases were seen in private practice within a year. Search of the literature for the past decade reveals no specific report of this particular syndrome. These are cases of pos-

Read before the Texas Diabetes Association Fort Worth, Texas.

terior column involvement in which no other etiologic agent than long standing, poorly controlled diabetes has been found. When diabetic neuropathy affects only the spinal cord, the posterior columns of Goll and Burdoch are most frequently involved. Disturbed sensation, mostly loss of vibration sense, ataxia, areflexia were not late residual signs and symptoms of peripheral neuritis, as there had never been pain, and there was no peripheral nerve tenderness, distribution, or thickening. Treatment with adequate control of the hyperglycemic state, massive doses of vitamins, parenterally and orally, including the B fractions, vitamin E, and crude liver extract has over a period of 8 and 10 months produced gratifying but slow improvement.

Case Reports

Case 1. W.C.M., a white male, age 48, was first seen in November 1948 with the complaints of ready fatiguability, clumsiness in dressing himself and other fine coordinated movements, occasional stumbling, "tightness in the legs," slower motion than formerly, a tendency toward being anti-social, increasing nervousness, libido loss and diminished mental alacrity, all of 11 months' duration, getting worse. He was a known diabetic for 17 years. In the past year he had been on a 2200 calorie diet and 40 units protamine zinc insulin with 10 units regular insulin doses before breakfast and before supper. He had never been in coma but in the past 2 years had suffered numerous alarming insulin shock states. Health otherwise had previously been good. His mother had died at the age 65, also diabetic. Positive physical findings were confined to the central nervous system. He walked with a wider than usual broad base. There was unsteadiness and staggering with quick turns and ambulatory maneuvers. Romberg test was positive. Use of his eyes directed to the ground was obligatory for walking. All tests for ataxic motion were positive. Coordination was poor. There was loss of vibratory sensation in the lower extremities. Position sense was absent. Both the patellar and achilles tendon reflexes were absent and sluggish elsewhere.

Laboratory data was as follows: Erythrocytes 4,890,000, hemoglobin 14.8 grams, leukocytes 8,550, polymorphonuclears 58, stab cells 7, lymphocytes 25 monocytes 2, eosinophils 8. All indices were within normal limits. Blood serology was negative. Urinalyses were normal. Sedimentation times was 7 mm. in one hour. Non-protein nitrogen was 40.2 mgm. %. Creatinine was 1.5 mgm. %. Glucose tolerance curve was of the diabetic type. Serum calcium, phosphorous, sodium, potassium, chlorides, cholesterol, and uric acid were all within normal limits. Spinal fluid chemistry, serology and dynamics were normal. Sternal puncture revealed normal bone marrow cytology. Gastric analysis was normal. Electrocardiogram was normal. Basal metabolism revealed plus 3%. X-Ray of the chest was normal. Barium meal revealed no gastro-intestinal abnormality. Stool specimens were negative for occult blood. Neurological consultation supported a diagnosis of posterior column disease.

Following a year's treatment with large doses of multi-vitamins and liver extract, parenterally administered, and stricter control of his diabetes, this patient has made an almost complete recovery.

Case No. 2. J.E.B., a white male, age 60, was first seen September, 1948, because of weight loss, weakness, breathlessness, stumbling, and falling to one side or another frequently. Diabetes was first diagnosed in 1941, since which time he was poorly controlled with inadequate insulin dosage, no dietary instruction. There was no history of ketosis. Initial examination disclosed a marked microcytic, hypochromic anemia, tarry stools, hepatomegaly, hypertension, albuminuria, mass in the right lateral mid-abdomen, uncontrolled diabetes with acetonuria, and a suppurative cellulitis of the right foot following a puncture wound. Barium enema revealed a filling defect in the mid-cecum, and a grade I adenocarcinoma of the cecum was removed. Post-operative convalescense was uneventful, blood count returned to normal, liver size diminished, the infection of the foot gradually cleared, and the patient was controlled well with diet and 35 units protamine zinc insulin and 10 units regular insulin before breakfast. Hypertension persisted. One month after operation and correction of anemia, debility, and the uncontrolled diabetes, attention was directed to the neurologic state. The lower extremities revealed loss of vibration sense, absent deep tendon reflexes, loss of position sense, and a positive Romberg test. There was no nerve tenderness, no history of pain.

Laboratory data at this time was as follows: Erythrocytes 5,250,000, hemoglobin 16.2 gms., leukocytes 6,600, differential percentage normal. All indices were within normal limits. Blood serology was negative. Urinalyses were normal. Glucose tolerance curve was typically diabetic. Fasting non-protein nitrogen was 40 mgm.%. Sedimentation rate was 8 mm. in one hour. Serum calcium, phosphorus, uric acid, cholesterol and chlorides were all within normal limits. Spinal fluid chemistry, serology and dynamics were normal. Sternal bone marrow revealed normal hematopoeisis. Gastric analysis revealed marked hypochlorhydria with poor response to histamine. The electrocardiogram revealed left ventricular hypertrophy and strain. Basal metabolic rate was normal. Post-operative barium enema revealed good functioning anastomosis (terminal ileum to mid-transverse colon). Liver function tests were normal. Neurological consultation agreed with a diagnosis of posterior column disease.

Following a year's intensive therapy consisting of better control of the diabetic state, parenteral

multi-vitamins and liver extract, this patient has made an approximate 50% recovery from his neuropathic complication. Hypertension and periodic moderate albuminuria persist. To date there is no sign of recurrence of his malignancy. The liver is still enlarged but compensated. Repeated peripheral blood studies have failed to hint of a primary macrocytic anemia. Reticulocyte count has never been over 3%.

Since these two cases were seen, every diabetic patient now studied receives an initial and periodic complete neurological survey. More attention to this system will undoubtedly reveal a higher incidence of neuropathies and, more important, detect this complication in its incipiency, where therapy might produce better results.

Summary

1. Two cases of involvement of the posterior columns of the spinal cord in uncontrolled diabetics are reported. No other etiologic factor except diabetes was found.
2. Response to therapy, consisting of better control of the diabetic state, parenterally administered multi-vitamins and liver extract in large doses, has been gratifying thus far.
3. No attempt has been made to shed further light on the patho-physiology of this interesting form of diabetic neuropathy. Present concepts have been reviewed.
4. A plea is made to direct more attention to the nervous system, initially and in periodic follow-ups, in all diabetics, thereby detecting neuropathies in the early phase, during which therapy may be more beneficial.

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Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. 261218

Presentation

A 19-year-old white student was brought to the hospital unconscious at 7 P.M., February 27, 1950, and died March 4, 1950, at 2:20 A.M. The patient was found unconscious at home 19 hours after he was last seen in good health. On the morning of the accident a fellow student stopped in front of the patient's house, intending to drive him to school. When no one appeared he went on his way but returned that evening and called the police when he found the house quiet. The patient was in the same room on the second floor as his mother and both were unconscious. All windows and doors were tightly closed and the house was pervaded by an odor of gas, strongest in the basement where a dead cat was found. An obstructed exhaust pipe from a gas-burning furnace was discovered later.

At the time he entered the ward the axillary temperature was 103° F. The patient was in deep coma. The lungs were clear, but respirations were shallow and rapid, and the rate was 60 per minute. The heart sounds were of fair quality with regular rhythm and a rate of 140. Blood pressure was not obtainable. The abdomen was normal. The left upper extremity was diffusely and tightly swollen with erythema and vesiculation; the upper arm showed the pattern of fingers of the right hand. The lower portion of the left forearm was deeply cyanotic, cold, and pulseless. Erythema and vesiculation also affected the left pectoral region and the right hand. Ankle clonus and bilateral extensor plantar responses were elicited. The other deep reflexes were hyperactive. The skin, abdominal, and cremasteric reflexes were absent. There was generalized flaccidity.

Red cells of the blood numbered 6.47 million per

cmm. on admission and 5.4 million the following day. Hemoglobin was 19 gm. per 100 cc. on admission and 17 gm. subsequently. The white cell count remained between 20 and 25 thousand with 80 per cent neutrophils. Catheterization yielded 900 cc. of urine which was normal; two days later 4 plus albumin and red and white cells were found in the sediment. The stool was guaiac negative. A Kahn test of the blood was negative. Blood urea nitrogen rose from 42 mg per cent March 1 to 145 two days later. Carbon dioxide combining power of the plasma remained near the level of 30 vol. per cent on March 1, 2, and 3. Serum chlorides on these dates were 512, 504, and 532 mg. per cent. An electrocardiogram on March 1 showed sinus tachycardia, rate 125; PR .19 second; low voltage QRS complexes in limb leads; T 1 flat; QT interval .28 second (upper normal for rate). This record was considered to be minimally abnormal. A sample of blood taken several hours after admission showed 10 per cent carbon monoxide saturation. An unsatisfactory chest film on March 1 suggested pulmonary congestion and edema.

Initially, oxygen with 10 per cent carbon dioxide and later oxygen by nasal catheter were administered and an airway was inserted. He was also treated with blood, plasma, fluids, and penicillin. Blood pressure rose to 135/105 several hours after admission, but coma continued. Cyanosis of the left forearm deepened to the point of impending gangrene and was unaffected by paravertebral block and abduction of the arm. These measures failed to restore pulsations in the extremity. Decreasing urinary output progressed to anuria on March 3. Increasing laboriousness and irregularity of respiration terminated in death at 2:20 A.M., March 4, 1950.

Differential Diagnosis

Dr. Charles D. Aring: The diagnosis of carbon monoxide poisoning is so obvious in this case—what with the cat dead in the cellar and the mother comatose on the second floor—that it seems what is wanted is not diagnostic speculation so much as a review of what happens to people who are poisoned with carbon monoxide and some attempt at delineation of the physiopathology and therapy.

After acute exposure to carbon monoxide a galaxy of symptoms develop including nausea, fullness or pain in the head, vertigo, weakness, and, with continued exposure, faintness, somnolence and coma. Finally loss of sphincter control and even convulsions may be noted. It is said to require from 30 to 50 per cent saturation of the blood to produce some of the more serious symptoms.

With chronic exposure persistent headache is a common phenomenon as is giddiness, weakness, impaired mentation, and at times psychotic states. It has been noted that workers in garages where the air is heavily contaminated are prone to suffer headache. When the intoxication is prolonged and severe, as it was in our patient, damaged neural structures result in palsies, anesthesias, tremors, and dysarthrias which in some instances have reminded of multiple sclerosis. Neurotic states are said to occur although I have never seen them, and psychotic states are characterized by confusion, mania, and hallucinations.

Almost any neurological symptom may be expected and has been reported at one time or another in case to case. Spastic quadriplegia, speech defect, catatonia, and Korsakoff's syndrome are some evidence of the variance in the clinical range. During recovery from the acute phase, a rather characteristic sign is painful spasms which occur in the skeletal muscles. It is interesting in some cases where recovery seemed to be complete that relative health for days or weeks has been followed by residual signs. Some patients seemingly well after exposure have come down with one or another chronic neurological syndrome. The clinical syndromes most often reported in survivors are protracted coma—in my experience the most common—psychosis, parkinsonism, polyneuritis, blindness, and varied asthenic states.

In our patient we see that a sample of blood taken several hours after admission to the hospital showed 10 per cent carbon monoxide saturation. This would seem to be evidence of severe exposure since carbon monoxide is rapidly eliminated from the blood. Within an hour usually half is gotten rid of and all traces are said to be gone four hours after removal from exposure. Critical carboxyhemoglobin level is said to be about 50 per cent of saturation and 75 per cent to be immediately fatal. It is interesting that levels of 30 per cent have been reported in those exposed to traffic fumes for eight hours. I'll leave the remainder of the laboratory discussion for Doctor Dutra.

In carbon monoxide poisoning the main lesions are in vessels within the nervous system as well as without. The parenchyma

of the central nervous system is severely involved. This will be discussed in some detail later. The peripheral nervous system is also affected. It is invariably the case that the nerves supplying the limbs on which the patient is found to be lying are the most often affected. In our patient it would seem that the heart, lung, and kidney were severely involved and that death was associated with kidney failure.

Now a word about the physiopathology. As you know, carbon monoxide has a potent affinity for hemoglobin, said to be 200 to 400 times that of oxygen. The fact that hemoglobin combines so readily with carbon monoxide results in local asphyxia of tissues. In the central nervous system it has seemed as though there were some specificity of the involvement. In most reports particular predilection seemed to be for white matter. There is also predilection for certain of the basal ganglia, the globus pallidus particularly, and in the cortex the 3rd and 4th cortical layers are particularly involved. In the cases that we have seen, the involvement has been predominantly in white matter.

It has been wondered whether or not carbon monoxide acts in other ways besides by asphyxia. There is little evidence that such is the case. While the analogy isn't particularly pertinent, 50 per cent saturation with carbon monoxide produces a graver clinical syndrome than that of a patient with anemia who has 50 per cent reduction in his hemoglobin for whatever the cause. On these grounds as well as others it has been thought that carbon monoxide is a direct tissue poison, that it poisons the nerve cells directly and other tissues as well. There is evidence too that the effect on blood vessels and especially on vascular endothelium is responsible for much of the damage seen in many tissues, particularly in the central nervous system. While the hypoxia surely is detrimental to vascular endothelium, the effects secondary to this may be of a vasoparalytic nature with occlusion, edema, and perivascular hemorrhage.

Various factors have been pointed to in attempts to explain the predilection of carbon monoxide for certain structures in the central nervous system. Bilateral softening of the globus pallidus has been seen not infrequently. The globus pallidus is supplied with blood rather uniquely when compared with other grey masses. The vessels from the middle cerebral artery supplying the globus pallidus are small and enter this nucleus at a sharp angle and pursue a recurrent course, so that there are several areas of impedance to flow. The reason given for predominant involvement in the third and fourth cortical layers is that the fine caliber of arterioles and venules in this particular part of the cortex favors occlusion. It is known that in experimental lead poisoning and in cerebral malaria comparable changes may be found in the third and fourth cortical layers, presumed to be on the basis of this fine vascular network. The sub-cortical white matter, which is predominantly involved in almost every case of severe exposure to carbon monoxide contains a relative sparsity of anastamosis.

It is interesting that experimental sublethal potassium cyanide poisoning and experimental capillary fat embolism (Hurst[1]) result in lesions almost entirely confined to white matter. This has been explained by the sparsity of anastamoses in the white matter as compared to gray. It appears that with repeated sub-lethal doses of a noxious agent the cortex can survive while the white matter cannot. It requires a rather severe deprivation of blood supply to destroy cortical cells.

In summary, I would expect widespread parenchymal involvement of the brain. Scar formation is possible since the patient lived for about seven days after exposure to carbon monoxide. Some hyalin and fatty changes may be found in vascular coats with diapedesis and thrombosis. I have mentioned the areas of predilection where we expect to find cerebral lesions, the white matter particularly, the globus pallidus, 3rd and 4th cortical layers, but changes have been reported in every part of the central nervous system from case to case. The muscles and peripheral nerves must be involved in limbs that bore the body weight. One would expect also widespread toxi-degenerative changes in the other organs; the cardiac muscle and lungs particularly were affected

in our patient, and the kidneys must have been heavily involved.

While this isn't the place to discuss treatment, it is worth mentioning that treatment seemed adequate in our patient. I call your attention to an article that appeared two years ago on the use of intravenous procaine hydrochloride in the treatment of asphyxia due to carbon monoxide.[2] Several cases were discussed with electroencephalographic records before and after the administration of procaine. These persons had been severely exposed to carbon monoxide and were brought to the hospital usually in a coma or with some severe neurological manifestations. They were reported to show considerable improvement in the clinical state and electroencephalogram after the use of procaine. The rationale for procaine therapy is stated as the analogy between cerebral asphyxia and local asphyxia elsewhere as seen in embolism, toxic or reflex anuria, Raynaud's disease, frostbite, and other gangrenous conditions in which procaine has seemed to be helpful. Examining the case reports and collating our own material has not allowed us to feel hopeful about this attempt at therapy.

*Dr. Robert A. Helm**: We have tried intravenous procaine in five or six patients with carbon monoxide poisoning since the appearance of the article on this subject. There was no noticeable beneficial effect in any of them including this patient; some recovered but I don't think it was related to the procaine, and those who did not get well were not benefited, even temporarily.

Dr. Eugene B. Ferris: In this case the downhill course was steady which the procaine didn't alter.

Dr. William Ransohoff: I wonder if Dr. Aring will comment on the apparent recovery and then relapse seen in some patients. I recall one patient whom we treated here for carbon monoxide poisoning who was sent home in ten days and came back about ten days later with neurological signs and went on to die.

Dr. Aring: I have no idea why this happens occasionally. I have never seen it myself.

*Chief Medical Resident.

Dr. Ferris: Dr. Brown, could you tell us about the treatment routine in the Receiving Ward?

Dr. Josiah Brown: Usually when a patient comes to the Receiving Ward he has been treated for one-half to one hour with carbon dioxide and oxygen by the Life-Saving Squad. Sometimes carbon dioxide and oxygen is continued for a while in the Receiving Ward. Sometimes these patients are switched to oxygen alone. Most regain consciousness and are sent out; those who do not are admitted to the hospital.

Dr. Ferris: During the war years Drs. Henry Ryder, George Engel, and I did some studies on carbon monoxide levels in the blood in relation to the use of fire extinguishers in closed spaces. We built up carbon monoxide level in normal controls to about 20 to 25 per cent with few, if any, symptoms except very slight headache. So it is easy to see why people can run around with considerable carbon monoxide in the blood without having any particular symptoms.

Dr. Aring mentioned the difference between the pathology and the symptomatology of anemia with 50 per cent hemoglobin and carbon monoxide poisoning with 50 per cent ineffective hemoglobin due to carbon monoxide hemoglobin. The corollary is not a particularly good one in terms of suggesting some specific tissue change because we know that with anemia there is, in association with it, a compensatory increase in cardiac output and blood flow which almost makes up for the deficit. In other words, the increased turnover of the hemoglobin present almost makes up for the actual reduction in hemoglobin so that one does not get much tissue anoxia in chronic anemia unless it is very severe.

Discussion of Pathology

*Dr. Frank R. Dutra**: The body was that of a well-nourished and well-hydrated young man. The fact that he was dehydrated when he was admitted to the hospital is indicated by the high hemoglobin value at that time; this was down to normal on the day after admission, following intravenous fluids. Most

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patients admitted in coma from carbon monoxide poisoning are severely dehydrated, and this is probably partly responsible for the fever observed at this time.

There was a large area of erythema and edema of the tissues of the left breast, and the left forearm was swollen and marked with excoriations of the epidermis, the remains of the blisters that he had had at the time of admission. The blisters had ruptured and the dermis was covered with crusts in these areas. I would like to digress for a moment to state that such blisters, in my opinion, are the result of a diminution in the flow of the blood to the area, coupled with the fact that the blood is carrying suboptimal amounts of oxygen. This combination results in a form of "pressure sore" because of deficiency of oxygen supply to the skin. In the present case, the epidermis of the left forearm and the left breast showed necrosis, which is part of the damage from the anoxia. Incidentally, the large vessels in the arm were not damaged, although there were thrombi in some smaller veins of the skin and subcutaneous fat.

The heart weighed 410 grams, which is somewhat larger than average for a lad of this size, and it was markedly dilated. There was diffuse hemorrhage throughout the outer half of the myocardium of the left ventricle, while the inner half of the muscle showed only diffuse degenerative changes. There were also focal areas of hemorrhage and necrosis in the papillary muscles of the left ventricle; this is a fairly characteristic lesion in carbon monoxide poisoning even when there is no other demonstrable damage to the heart.

The lungs were only slightly heavier than normal, and they showed a few small foci in which there was exudate of polymorphonuclear leukocytes. There were some hemorrhages, and there were some focal areas of atelectasis. This is a real tribute to the house officers who took care of this patient because it is nearly a certainty that he would have died of pneumonia within a few days after admission if he had not been given appropriate antibiotic therapy and ancillary care. We are all convinced that patients in coma from carbon monoxide poisoning are now much more likely to recover because of antibiotics and chemotherapy to combat infections.

The liver and spleen were large, due to passive hyperemia and parenchymatous degeneration, and there was lower nephron nephrosis.

There was some pigment in these kidneys and this was probably myoglobin released from the necrotic pectoralis major muscle and from the muscles of the left arm which were also necrotic. However, I believe that pigment is not necessary for the development of lower nephron nephrosis, and that this lesion can occur simply from the tissue oxygen deficiency.

The brain weighed 1725 grams, and it was swollen and edematous with diffuse hemorrhages especially throughout the white matter. There were no focal lesions in the globus pallidus, but the white matter of the cerebrum and the cerebellum were badly damaged.

The left arm and breast examined on the second hospital day showed erythema and blisters that are often mistakenly identified as burns, but which are actually caused by the pressure. Viewing a cross section through the breast, in one area there is normal skin, the muscle fibers are well preserved, the bundles appear normal, and the fat is lobulated and appears normal. In another area the fat is hemorrhagic, there are hemorrhages in the skin, and the muscle is pale, friable, necrotic tissue.

In a cross section of the heart a hemorrhage is seen limited to the outer layer of the myocardium of the left ventricle. There were degenerative phenomena in the myocardial fibers and the heart is dilated.

A gross representation of the lower nephron nephrosis showed the bluish red hyperemic pyramids and the relatively less congested but nevertheless hyperemic cortex, and there is certainly some swelling of the cortex.

In the brain hemorrhages are prominent in all of the white matter, including corpus callosum, and there is relatively little damage in the region of the basal ganglia. A photomicrograph shows the way these hemorhages are located perivascularly, extending out into the adjacent tissues with softening of white matter. In a gross section of

the cerebellum it is to be noted that there are smaller perivascular hemorrhages in the white matter.

Microscopic examination of a section of the skin from the left side of the chest shows thrombi in some of the smaller vessels. From left to right one sees the normal epidermis, and then it becomes necrotic, and finally it is actually absent. You can see that there are hemorrhages beneath the area of epidermal necrosis.

In the sections of the pectoralis major muscle there was diffuse coagulation necrosis with hemorrhages into the fat and between the muscle bundles. The fibers were washed out and there were infiltrating inammatory cells; small vessels contained thrombi.

In the wall of the left ventricle there was diffuse hemorrhage with myocardial fibers still preserved and not completely necrotic. The papillary muscles contained areas of ischemic necrosis and hemorrhage.

There was atelectasis and focal hemorrhage in the lung but practically no cellular exudate. There were some large mononuclear cells and occasional polymorphonuclear leukocytes but no active purulent inflammation.

In the kidney one of the tubules was completely blocked with granular pigment. There is degeneration of the epithelium lining these tubules and many of the tubules contain pigment and are undergoing degeneration. There is actually rupture of tubules in some places, so there is no question but that this represents a lower nephron nephrosis. In the convoluted tubules of the cortex there is severe parenchymatous degeneration of the convoluted tubules with casts in the lumens.

In the myelin sheath stains of the brain there were local perivascular hemorrhages and diffuse demyelinization in the white matter. The dentate nucleus of the cerebellum contained cells with nuclei eccentrically placed and with clear central cytoplasm. Where processes were visible, they were thickened and swollen. There were ghost cells, cells that were actually disappearing because they had become devitalized. There were degenerative phenomena in the ganglion cells throughout the brain.

I want to compliment Dr. Aring on his discussion of the phenomena that make carbon monoxide poisoning the serious condition that it is. The affinity that hemoglobin has for carbon monoxide is so great that as little as 0.5 per cent in the atmosphere will be quickly fatal and as little as 0.1 per cent can be fatal over a longer period of time. The carbon monoxide tends to accumulate in the blood at a slower rate at these lower concentrations but finally it may reach a lethal concentration.

In carbon monoxide poisoning there is also an increased affinity of the hemoglobin for the *oxygen* that it contains. Oxyhemoglobin will dissociate when the oxygen tension in the plasma falls to a certain level. If the tissue and plasma oxygen tension reaches a level of 28 mm. mercury, half of the molecules of oxyhemoglobin will dissociate, and the oxygen will go into the tissues, but if there is a carbon monoxide molecule in the place of some of these oxygen molecules, the bond between the hemoglobin and the other oxygen molecules increases in strength, so that it requires a greater degree of oxygen deficiency in the tissues before oxygen is liberated from its combination with hemoglobin. When 40 per cent of the hemoglobin is combined with carbon monoxide, the tissue oxygen tension must fall to 12 mm. mercury in order that half of the potentially available oxygen be given up to the tissues.

Another factor of great importance in evaluating a patient's condition is the duration of coma. If a man is exposed to an atmosphere containing 0.5 per cent carbon monoxide, his blood carbon monoxide will go up rather fast, and if he is rescued from the contaminated atmosphere when 50 per cent of his hemoglobin has been converted to carboxyhemoglobin, he will recover quickly and the blood will be restored to normal, if he be treated with inhalation of "carbogen" (oxygen 95% and carbon dioxide 5%). Such a man will suffer no aftereffects. But if one is exposed to an atmosphere containing carbon monoxide at a concentration of 0.1 per cent, the equilibrium of which is about 50 per cent carboxyhemoglobin (i.e., after a long exposure his blood saturation will be 50 per cent, and then will

stay at that 50 per cent indefinitely because this is the point at which the amount of carbon monoxide going into the blood is the same as the amount of carbon monoxide coming out of the blood). This man, whose blood vessels are gradually relaxing and losing fluid, followed by diapedesis of erythrocytes, is also rescued, but after 24 hours of deep coma. This was the situation with this boy; he had severe oxygen deficiency for a long period, so that we might well expect resuscitation to be difficult. I think that the increased affinity of hemoglobin for carbon monoxide, the strengthening of the bonds between hemoglobin and oxygen, and the duration of the oxygen deficiency can explain everything that we know about carbon monoxide poisoning.

Clinical Diagnosis

Carbon monoxide poisoning.

Dr. Aring's Diagnosis

Carbon monoxide poisoning.

Anatomical Diagnosis

Acute carbon monoxide anoxemia.

Edema of brain.

Focal necrosis, myocardium of left ventricle.

Hemorrhages of myocardium.

Lower nephron nephrosis.

Parenchymatous degeneration of myocardium, brain, liver, adrenal glands, renal tubules, testes, skeletal muscles (left pectoralis major, muscles of left forearm, and rectus abdominus).

Pulmonary atelectasis, focal, bilateral.

Pulmonary hemorrhage, focal, bilateral.

Passive hyperemia of viscera.

Petechiae of brain, optic chiasm, gastric mucosa, mucosa of urinary bladder, thymus.

Erosion of mucosa of stomach.

Necrosis of epidermis, right hand, left breast, and left forearm.

Chemosis of left conjunctiva.

Dr. Charles Kiely: Since two people were exposed for the same length of time, why did one die and the other not?

Dr. Dutra: The other had approximately the same exposure. The fact that this boy survived in the hospital for 7 days indicates that he was severely poisoned but was close to survival. This exposure certainly was near the LD_{50}, since one survived and one died.

Dr. Edward Gall: Did you indicate, Dr. Dutra, what the carbon monoxide content of illuminating gas was?

Dr. Dutra: In Cincinnati we have no carbon monoxide in illuminating gas. It has been entirely natural gas for the past 10 years.

Dr. Ferris: Why are people said to die of illuminating gas poisoning?

Dr. Dutra: They do not die of natural gas poisoning. They are only *said* to die of natural gas poisoning.

I went out to the home where this boy was exposed. There was a furnace that had been converted to gas connected to a flue with an intake vent on the flue pipe. The flue connected with the chimney. This was one of those chimneys where the base of the chimney is right at a level where the flue comes in. This has real practical significance, for when one converts a coal furnace to a gas furnace, there is a lot of residue in the chimney that cannot be swept out. It is caked within the chimney. As the thermal conditions in the chimney are altered, this caked soot begins to crack and break loose, and may fill the base of the chimney. In this boy's home some bricks and concrete had fallen from the chimney wall and obstructed the flue orifice, so that the gases of combustion were passing out of the intake vent in the side of the flue and infiltrating the whole house.

Now why are people killed by natural gas? They are killed because as natural gas burns the products of combustion include water, carbon dioxide, and even under the best of conditions some carbon monoxide is also produced.

Dr. Ferris: Dr. Dutra, is it an old wives' tale then that people turn on the gas and close the windows and die?

Dr. Dutra: In areas served by natural gas such deaths are not suicides. There are records of many cases where death was homicidal and an attempt was made to set the scene to look like suicide. A person does not die of "methane poisoning." All that methane can do is replace some of the oxygen in the air. In a closed room, in a new house, with, the best possible ordinary construction, there will be at least two or three changes of atmospheric air every hour. With such ventilation, depletion of oxygen could not occur. The same thing can be said for the utilization by the combustion process of the oxygen in the atmosphere; that does not cause death in closed rooms.

Dr. Ferris: Is methane inert like nitrogen?

Dr. Dutra: Yes. The only danger of unburned natural gas is the hazard of an explosion. That requires an atmosphere in which there is about 15 per cent natural gas.

Dr. Gall: Would you want to estimate what caused death in this patient? Do you believe the myocardial lesion was responsible?

Dr. Dutra: It is not possible to state whether he died of brain damage, or heart lesions, or as a result of the lower nephron nephrosis. He died of the residuals of carbon monoxide poisoning.

Dr. Ferris: Was there any hemorrhage in the wall of the right ventricle?

Dr. Dutra: There was no hemorrhage in the tissue of the right ventricle. The myocardial fibers show some degeneration.

Dr. Ferris: What about the pericardium?

Dr. Dutra: There were hemorrhages in the subepicardial connective tissues. That is a common phenomenon in asphyxia and oxygen deficiency, regardless of the cause of the deficiency.

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